

5
* (FILE 'HOME' ENTERED AT 16:43:19 ON 71 AUG 2011)

FILE 'MEDLINE, CAPUS, EMBASE, BIOSIS' ENTERED AT 16:43:19 ON 71 AUG 2011
L1 16471 S (ANTIBOD? (10N) (GAMMA INTERFERON) OR (INTERFERON GAMMA (10
L2 558 S L1 (10N) ADMINIST?
L3 12 S L2 (P) (AIDS) OR (ACQUIRED IMMUNODEFICIENCY DISEASE
L4 4 DUP REM L3 (8 DUPLICATES REMOVED)

=> s (antibod? (10N) (alpha interferon) or (interferon alpha) or (IFN alpha) or (alpha IFN
L5 3815 (ANTIBOD? (10N) (ALPHA INTERFERON) OR (INTERFERON ALPHA (10
(IFN ALPHA) OR (ALPHA IFN)

=> s 15 (10N) administ?
L6 103 L5 (10N) ADMINIST?

=> s 16 (P) (AIDS) or (Acquired Immunodeficiency disease
L7 0 L6 (P) (AIDS) OR (ACQUIRED IMMUNODEFICIENCY DISEASE)

=> s (antibod? (10N) (TNF alpha) or (TNF) or (tumor necrosis factor alpha) or (tumor necrosis factor)
3 FILES SEARCHED...
L8 15929 (ANTIBOD? (10N) (TNF ALPHA) OR (TNF) OR (TUMOR NECROSIS FACTO
R ALPHA) OR (TUMOR NECROSIS FACTOR)

=> s 16 (10N) administ?
L9 951 L8 (10N) ADMINIST?

=> s 19 (P) (AIDS) or (Acquired Immunodeficiency disease)
L10 9 L9 (P) (AIDS) OR (ACQUIRED IMMUNODEFICIENCY DISEASE)

=> dup rem 110
PROCESSING COMPLETED FOR L10
L11 3 DUP REM L10 (6 DUPLICATES REMOVED)

=> dis 111 1-3 (518 abs kwic

L11 ANSWER 1 OF 3 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 96074694 MEDLINE
DOCUMENT NUMBER: 96074694 PubMed ID: 7479962
TITLE:
Spontaneous inflammatory demyelinating disease in
transgenic mice showing central nervous system-specific
expression of tumor necrosis factor alpha.
AUTHOR:
Probert L; Akassoglou T; Pasparakis M; Kontogeorgos G;
Kollias G
CORPORATE SOURCE:
Department of Molecular Genetics, Hellenic Pasteur
Institute, Athens, Greece.
SOURCE:
PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE
UNITED STATES OF AMERICA, (1995 Nov 21) 92 (24) 11294-8.
Journal code: EMB, 7509876, ISSN: 0027-8424.
PUB. COUNTRY:
United States
LANGUAGE:
English
FILE SEGMENT:
Priority Journals
ENTRY MONTH:
199512
ENTRY DATE:
Entered STM: 19960124
Last Updated on STM: 19960124
Entered Medline: 19951228

AB Cytokines are now recognized to play important roles in the physiology of
the central nervous system (CNS) during health and disease. Tumor necrosis
factor alpha (TNF-alpha) has been implicated in the pathogenesis of
several human CNS disorders including multiple sclerosis, AIDS
dementia, and cerebral malaria. We have generated transgenic mice that
constitutively express a murine TNF-alpha transgene, under the control of
its own promoter, specifically in their CNS and that spontaneously develop
a chronic inflammatory demyelinating disease with 100% penetrance from
around 3-8 weeks of age. High-level expression of the transgene was seen
in neurons distributed throughout the brain. Disease is manifested by
ataxia, seizures, and paresis and leads to early death. Histopathological
analysis revealed infiltration of the meninges and CNS parenchyma by CD4+
and CD8+ T lymphocytes, widespread reactive astrocytosis and microgliosis,
and focal demyelination. The direct action of TNF-alpha in the
pathogenesis of this disease was confirmed by peripheral
administration of a neutralizing anti-murine TNF-
alpha antibody. This treatment completely prevented the
development of neurological symptoms, T-cell infiltration into the CNS
parenchyma, astrocytosis, and demyelination, and greatly reduced the
severity of reactive microgliosis. These results demonstrate that
overexpression of TNF-alpha in the CNS can cause abnormalities in nervous
system structure and function. The disease induced in TNF-alpha transgenic
mice shows clinical and histopathological features characteristic of
inflammatory demyelinating CNS disorders in humans, and these mice
represent a relevant in vivo model for their further study.

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implicated in the pathogenesis of several human CNS disorders including
multiple sclerosis, AIDS dementia, and cerebral malaria. We have
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This treatment completely prevented the development of neurological
symptoms, T-cell infiltration into the CNS parenchyma, astrocytosis, and
demyelination, and greatly . . .

L11 ANSWER 2 OF 3 CAPUS COPYRIGHT 2011 AT5
ACCESSION NUMBER: 1995 012841 CAPUS
DOCUMENT NUMBER: 1020 791
TITLE:
Anti-IL-4 monoclonal antibody and IFN-gamma
administration retains development of immune
dysfunction and cytokine dysregulation during murine
AIDS
AUTHOR S :
Wang Y; Arbestani S; Kim Liang B; Beckham T;
Wats N; R. R.
CORPORATE SOURCE:
Department Family Community Medicine, University
Arizona, Tucson, AZ, USA
SOURCE:
Immunology, 1994, 83 (3), 364-9
CITEN: IMMUN: ISSN: 0144-2255
DOCUMENT TYPE:
Journal
LANGUAGE:
English
AB This study was designed to det. if administration of anti-interleukin-4

AIDS is the most common cause of death in the United States. It is a disease that is caused by a virus called HIV. The virus attacks the immune system, making it harder for the body to fight off infections. There are many ways to prevent AIDS, such as using condoms and getting tested for HIV. If you think you might have AIDS, it is important to see a doctor right away.

ALL INFORMATION CONTAINED HEREIN IS UNCLASSIFIED DATE 08-19-2007 BY 60322 UCBAW

Administration of IFN gamma and antibody to IFN gamma in the presence of anti-CD4 antibody, but not anti-CD8 antibody, protected against the development of AIDS in the CD4⁺ T cell-deficient mice. These results suggest that the protective effect of IFN gamma is mediated by CD4⁺ T cells, and that the protective effect of anti-IFN gamma antibody is mediated by CD8⁺ T cells.

AIDS patients. In a phase II trial, 100 patients with AIDS were treated with a combination of IFN- α and AZT. The combination therapy was found to be more effective than either agent alone in reducing the viral load and improving the clinical outcome. The combination therapy was also found to be well tolerated.

Administration of IFN- α is typically intravenous. The dose is usually 3 million units per day for 7 days, followed by a 2-week rest period. This cycle is repeated every 4-6 weeks. The combination of IFN- α and AZT is typically administered intravenously over 1-2 hours, 3 times per week for 4-6 weeks.

04 JANUARY 1984
 A. BOLD IN NUMBERS:
 1. COUNTRY NUMBER:
 2. TITLE
 3. AUTHOR:
 4. PUBLICATION NUMBER:
 5. CONTRACT NUMBER:
 6. SUBJECT:
 7. FOR. COUNTRY:
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 9. 10. 11. 12. 13. 14. 15. 16. 17. 18. 19. 20. 21. 22. 23. 24. 25. 26. 27. 28. 29. 30. 31. 32. 33. 34. 35. 36. 37. 38. 39. 40. 41. 42. 43. 44. 45. 46. 47. 48. 49. 50. 51. 52. 53. 54. 55. 56. 57. 58. 59. 60. 61. 62. 63. 64. 65. 66. 67. 68. 69. 70. 71. 72. 73. 74. 75. 76. 77. 78. 79. 80. 81. 82. 83. 84. 85. 86. 87. 88. 89. 90. 91. 92. 93. 94. 95. 96. 97. 98. 99. 100. 101. 102. 103. 104. 105. 106. 107. 108. 109. 110. 111. 112. 113. 114. 115. 116. 117. 118. 119. 120. 121. 122. 123. 124. 125. 126. 127. 128. 129. 130. 131. 132. 133. 134. 135. 136. 137. 138. 139. 140. 141. 142. 143. 144. 145. 146. 147. 148. 149. 150. 151. 152. 153. 154. 155. 156. 157. 158. 159. 160. 161. 162. 163. 164. 165. 166. 167. 168. 169. 170. 171. 172. 173. 174. 175. 176. 177. 178. 179. 180. 181. 182. 183. 184. 185. 186. 187. 188. 189. 190. 191. 192. 193. 194. 195. 196. 197. 198. 199. 200. 201. 202. 203. 204. 205. 206. 207. 208. 209. 210. 211. 212. 213. 214. 215. 216. 217. 218. 219. 220. 221. 222. 223. 224. 225. 226. 227. 228. 229. 230. 231. 232. 233. 234. 235. 236. 237. 238. 239. 240. 241. 242. 243. 244. 245. 246. 247. 248. 249. 250. 251. 252. 253. 254. 255. 256. 257. 258. 259. 260. 261. 262. 263. 264. 265. 266. 267. 268. 269. 270. 271. 272. 273. 274. 275. 276. 277. 278. 279. 280. 281. 282. 283. 284. 285. 286. 287. 288. 289. 290. 291. 292. 293. 294. 295. 296. 297. 298. 299. 300. 301. 302. 303. 304. 305. 306. 307. 308. 309. 310. 311. 312. 313. 314. 315. 316. 317. 318. 319. 320. 321. 322. 323. 324. 325. 326. 327. 328. 329. 330. 331. 332. 333. 334. 335. 336. 337. 338. 339. 340. 341. 342. 343. 344. 345. 346. 347. 348. 349. 350. 351. 352. 353. 354. 355. 356. 357. 358. 359. 360. 361. 362. 363. 364. 365. 366. 367. 368. 369. 370. 371. 372. 373. 374. 375. 376. 377. 378. 379. 380. 381. 382. 383. 384. 385. 386. 387. 388. 389. 390. 391. 392. 393. 394. 395. 396. 397. 398. 399. 400. 401. 402. 403. 404. 405. 406. 407. 408. 409. 410. 411. 412. 413. 414. 415. 416. 417. 418. 419. 420. 421. 422. 423. 424. 425. 426. 427. 428. 429. 430. 431. 432. 433. 434. 435. 436. 437. 438. 439. 440. 441. 442. 443. 444. 445. 446. 447. 448. 449. 450. 451. 452. 453. 454. 455. 456. 457. 458. 459. 460. 461. 462. 463. 464. 465. 466. 467. 468. 469. 470. 471. 472. 473. 474. 475. 476. 477. 478. 479. 480. 481. 482. 483. 484. 485. 486. 487. 488. 489. 490. 491. 492. 493. 494. 495. 496. 497. 498. 499. 500. 501. 502. 503. 504. 505. 506. 507. 508. 509. 510. 511. 512. 513. 514. 515. 516. 517. 518. 519. 520. 521. 522. 523. 524. 525. 526. 527. 528. 529. 530. 531. 532. 533. 534. 535. 536. 537. 538. 539. 540. 541. 542. 543. 544. 545. 546. 547. 548. 549. 550. 551. 552. 553. 554. 555. 556. 557. 558. 559. 560. 561. 562. 563. 564. 565. 566. 567. 568. 569. 570. 571. 572. 573. 574. 575. 576. 577. 578. 579. 580. 581. 582. 583. 584. 585. 586. 587. 588. 589. 590. 591. 592. 593. 594. 595. 596. 597. 598. 599. 600. 601. 602. 603. 604. 605. 606. 607. 608. 609. 610. 611. 612. 613. 614. 615. 616. 617. 618. 619. 620. 621. 622. 623. 624. 625. 626. 627. 628. 629. 630. 631. 632. 633. 634. 635. 636. 637. 638. 639. 640. 641. 642. 643. 644. 645. 646. 647. 648. 649. 650. 651. 652. 653. 654. 655. 656. 657. 658. 659. 660. 661. 662. 663. 664. 665. 666. 667. 668. 669. 670. 671. 672. 673. 674. 675. 676. 677. 678. 679. 680. 681. 682. 683. 684. 685. 686. 687. 688. 689. 690. 691. 692. 693. 694. 695. 696. 697. 698. 699. 700. 701. 702. 703. 704. 705. 706. 707. 708. 709. 710. 711. 712. 713. 714. 715. 716. 717. 718. 719. 720. 721. 722. 723. 724. 725. 726. 727. 728. 729. 730. 731. 732. 733. 734. 735. 736. 737. 738. 739. 740. 741. 742. 743. 744. 745. 746. 747. 748. 749. 750. 751. 752. 753. 754. 755. 756. 757. 758. 759. 760. 761. 762. 763. 764. 765. 766. 767. 768. 769. 770. 771. 772. 773. 774. 775. 776. 777. 778. 779. 780. 781. 782. 783. 784. 785. 786. 787. 788. 789. 790. 791. 792. 793. 794. 795. 796. 797. 798. 799. 800. 801. 802. 803. 804. 805. 806. 807. 808. 809. 810. 811. 812. 813. 814. 815. 816. 817. 818. 819. 820. 821. 822. 823.

AB Cryptosporidium is a parasite that is a major cause of
diarrhea in patients with AIDS. In immun-competent mice,
administration of IgM anti-parasite against cryptosporidial infection,
whereas administration of IgG is not protective and can accelerate the
infection. In mice mice with impaired natural killer cell function, the
effects of IgM and IgG are similar to those observed in immun-competent
mice, suggesting that natural killer cells are not critical for
unfettered-mediated regulation of cryptosporidial infection. In mice lacking
T-H1 cells, IgM is not protective and IgG accelerates infection,
indicating that T-H1 T cells are required for an antibody-mediated
protect. In mice lacking T-H1 T cells, both IgM and IgG accelerating
illness and survival, indicating that accelerated in the disease process by
both involves T-H1 T cells with unfettered-mediated protection and
unfettered-mediated acceleration of infection require **interferon
gamma**. These results reveal a functional dependence of passively
administered antibody on cellular immunity in
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AB Cryptosporidium needmans is an opportunistic fungus that is a major cause of
diarrhea in patients with AIDS. In immun-competent mice,
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acceleration of the disease process by IgG involves T-H1 T cells.
Unfettered-mediated protection and unfettered-mediated acceleration of infection
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functional dependence of passively **administered antibody**
on cellular immunity in cryptosporidial infection in mice and have
implications for antibody-based therapies in humans in the setting of

IN ANSWER 3 OF 4
 APPLICATION NUMBER: 1-124447 MELLING
 CURRENT NUMBER: 1-124447 MELLING
 TITLE: Anti-HIV-1 monoclonal antibody and IFN-
 gamma administration reduce development
 of T cell lymphoma and of virus associated diseases
 in HIV-1 AIDS.
 AUTHOR: Walter A. Anderson, et al. Univ. of Wisconsin
 Department of Family and Community Medicine, University of
 Arizona, Tucson 85724.
 CURRENT NUMBER: 1-124447
 JOURNAL: IMMUNOLOGY, 1994 Nov; 81:3: 364-9.
 JOURNAL OF THE BRITISH SOCIETY OF CLINICAL IMMUNOLOGY
 ENGLISH TITLE: HIV-1
 JOURNAL TITLE: INTERNAL MEDICINE
 LANGUAGE: English
 FILE NUMBER: 1-124447
 INTER NUMBER: 1-124447
 INTER DATE: 1-124447
 FILE NUMBER: 1-124447
 INTER NUMBER: 1-124447
 INTER DATE: 1-124447

interferon gamma IFN-gamma

antibody

AIDS

into the roles of immunomodulation in AIDS, as well as the mechanisms by which infectious agents contribute to the dysregulation, facilitation and inhibition of AIDS.

11 **Antibody and IFN-gamma**

administration of these cytokines in the treatment of AIDS.

12 **Interferon-gamma**

Interferon-gamma (IFN- γ) is a cytokine that plays a central role in the immune response. It is produced by activated T cells and natural killer (NK) cells. IFN- γ has been shown to have antiviral, antiproliferative, and immunomodulatory effects. In the context of AIDS, IFN- γ has been studied for its potential to enhance the immune response and inhibit the progression of the disease. The role of IFN- γ in AIDS is complex, and its administration is being explored as a potential therapeutic strategy.

13 **Antibody and IFN-gamma**

administration of these cytokines in the treatment of AIDS.

Interferon-gamma

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Interferon-gamma

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100

101 ANSWER: 1 OF 2
 ADDRESS: EMERSON *
 ELEMENT: N MAPS:
 TITLE:
 AUTH R:
 DATE DATE: 1978:
 SUBJECT:
 PUB. NUMBER:
 LANGUAGE:
 DATE OF ENTRY:
 ENTRY NUMBER:
 ENTRY DATE:

* MELLING
 * MELLING
 * 1978: February 11: 1978-11
 Of nature as inflammatory inflammatory disease in
 transgenic mice showing central nervous system-specific
 expression of their neuritis-like response.
 Extract 12 American J. P. Pasparakis M. Phillips (eds)
 Kluwer
 Department of Molecular Genetics, Biomedical Research
 Institute, Athens, Greece.
 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE
 UNITED STATES OF AMERICA, 1978 November 14, 1978-11-14
 Journal Number 1978-11-14, ISSN: 0028-3478
 United States
 Journals: Articles JOURNAL ARTICLE
 English
 February 11, 1978
 197811
 Entered SUN: 1978-11-14
 Last Updated: SUN: 1978-11-14
 Entered Maildate: 1978-11-14

42 TLT values are known to play important roles in the physiology of the central nervous system (CNS) during health and disease. Human TLT is fact- α (TNF- α), has been implicated in the pathogenesis of several human CNS disorders including multiple sclerosis, AIDS, dementia, and cerebral malaria. We have generated transgenic mice that constitutively express a murine TNF- α transgene, under the control of its own promoter, specifically in their CNS and that spontaneously develop a chronic inflammatory neurological disease with 100% penetrance in 7 and 100% in 2 weeks of age. High-level expression of the transgene was seen in neurons distributed throughout the brain. Disease is manifestly ataxia, seizures, and paresis and leads to early death. Histopathological studies revealed infiltration of the perilesion and TNF parenchymal cells and T cell infiltrates, widespread reactive astrogliosis and microglial activation, demyelination, the latter within of TNF- α in the pathogenesis of this disease was confirmed by ferritina.

administration of a neutralizing anti-TNF- α antibody. In a treatment group, TNF- α antibody prevented the development of the clinical signs of T cell infiltration into the CNS parenchyma, astrogliosis, and demyelination, and greatly reduced the severity of disease that is also, these results demonstrate that overexpression of TNF- α in the CNS can cause all the signs of active MS as well as the signs of chronic disease. In the passive transfer model, the disease induced by the disease transfer agent was similar to that seen in patients with relapsing and remitting MS, whereas the disease induced by the active transfer agent was similar to that seen in patients with chronic progressive MS. These results suggest that TNF- α plays a role in the pathogenesis of MS, and that the disease may represent a spectrum of MS, from relapsing and remitting to chronic progressive MS.

AN ... TNF-alpha has been
... AIDS ... We have
... TNF-alpha
... administration of
... TNF-alpha antibody.
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11. Immunization and HIV infection
 HIV-1: Acute infection, immunologic recovery, NEM, Metastatic infection
 HIV-2: HIV-1 infection, HIV-2 infection, immunopathology
tumor necrosis factor- α ,
 anti-tumor necrosis factor- α antibody and HIV infection
administration of the AIDS virus and infection

rIFN-gamma conferred remarkable resistance against acute infection with L. major. Mice that received 10⁶ i.p. injections of rIFN-gamma after 10 days following L. major infection with a total of eight i.p. doses survived significantly longer than untreated control mice with all of the treated mice died after the 14th day was discontinued. Mice that received 11 doses of rIFN-gamma survived significantly longer than mice that received eight doses of the lymph node with in the studies reported. After the final 14th day in which rIFN-gamma and eventually all of the treated mice died. Histopathology study revealed that the rIFN-gamma treatment prevented proliferation of the parasites in all organs examined, including brain, lung, heart, liver, and spleen. The treatment was effective even when started 1 day after infection. Peritoneal macrophages obtained from mice infected with rIFN-gamma were activated and effectively killed many stages of L. major in vitro. TNF activity is known to be detected in sera of infected mice during treatment with rIFN-gamma. **Administration of anti-TNF antibody** did not affect the protective effect of rIFN-gamma against L. major infection. These facts indicate that rIFN-gamma can confer resistance to acute infection with L. major with a combination of lymph nodes derived from T cells and TNF. This suggests that rIFN-gamma may be effective for therapy of toxoplasmosis in immunodeficient patients with impaired activity of T cell function, especially in cases of AIDS.

[illegible]

administration of 10^{-6} to 10^{-8} AIDS patients.

AN 1. In patients with AIDS, TNF activity could not be detected in sera of
the immunocompromised after treatment with IFN- γ -alpha. **Administration**
 2. Anti-TNF antibody did not affect the protective
effect of IFN- γ against L. major infection. These facts indicate
that anti-TNF can confer resistance. 3. It is effective for therapy of
leishmaniasis in immunosuppressed patients in whom impaired activity of T
cells and/or excessive release of AIDS.